early event in the angiogenic response that liberates cells for subsequent mobilization. In the present study, both PA and LPA weakly induced the chemotactic migration of endothelial cells from an established monolayer. The chemotactic response induced by PA and LPA was similar in intensity to that observed with optimal levels of the known protein endothelial cell chemoattractants, basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF). A markedly greater chemotactic response was effected by nanomolar concentrations of S1P, indicating that this platelet-derived factor plays an important role in a key aspect of angiogenesis, chemotactic migration of endothelial cells. The chemotactic response to S1P was completely inhibited by preincubation of endothelial cells with **antisense** oligonucleotides to the high-affinity S1P receptor, **Edg**-1. In addition, chemotaxis of endothelial cells to S1P was inhibited by preincubation of cells with specific inhibitors of tyrosine kinases, but inhibitors of phosphatidylinositol 3' kinase had little effect. Finally, LPA effectively stabilized endothelial monolayer barrier function, a late event in angiogenesis. Thus, the phospholipid growth factors, PA, S1P, and LPA, display divergent and potent effects on angiogenic properties of endothelial cells and angiogenic differentiation of endothelial cells potentially act in tandem to effectively induce neovascularization. These mediators may thus exert important roles in restoration of hematopoiesis, as they facilitate blood vessel formation at sites of transplanted stem cells, allowing the progeny of engrafted progenitors to move from marrow sinusoids to the peripheral vasculature.

2/3,AB/9 (Item 9 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10336813 99282821 PMID: 10354366
Lysophospholipid enhancement of human T cell sensitivity to diphtheria toxin by increased expression of heparin-binding epidermal growth factor.
Goetzl EJ; Kong Y; Kenney JS
Department of Medicine, University of California Medical Center, San Francisco 94143-0711, USA.
Proceedings of the Association of American Physicians (UNITED STATES) May-Jun 1999, 111 (3) p259-69, ISSN 1081-650X Journal Code: CDQ
Contract/Grant No.: HL31809, HL, NHLBI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The effects of lysophosphatidic acid (LPA) and sphingosine 1-phosphate (S1P) on T cell expression of heparin-binding epidermal growth factor-like growth factor (HB-EGF), the diphtheria toxin (DT) receptor, were investigated in the Tsup-1 cultured line of human CD4+ 8+ 3low T lymphoblastoma cells. Tsup-1 cells bear endothelial differentiation gene (**edg**)-2 and -4 encoded G protein-coupled receptors (GPCRs) for LPA and **Edg** -3 and -5 GPCRs for S1P. Suppression by DT of Tsup-1 cell protein synthesis was enhanced by LPA and S1P, with lipid structural specificity similar to that required for their recognition by **Edg** receptors. LPA and S1P increased the Tsup-1 cell level of immunoreactive HB-EGF, and neutralizing antibodies to HB-EGF inhibited LPA and S1P enhancement of Tsup-1 cell susceptibility to DT. Stabilized transfection of Tsup-1 cells with a combination of plasmids encoding **Edg**-2 plus -4 **antisense** mRNA suppressed the levels of **Edg**-2 and -4, but not **Edg**-3 and -5, in Western blots and reduced in parallel the increments in HB-EGF and susceptibility to DT evoked by LPA but not S1P. Similar transfection with **Edg**-3 plus -5 **antisense** plasmids suppressed Tsup-1 cell levels of immunoreactive **Edg**-3 and -5, but not **Edg**-2 or -4, and

2/3,AB/10 (Item 10 [redacted]om file: 155)
DIALOG(R)File 155:MEDLINE(R)
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10079834 99138900 PMID: 9973477
Lysophosphatidic acid and sphingosine 1-phosphate protection of T cells from apoptosis in association with suppression of Bax.
Goetzl EJ; Kong Y; Mei B
Department of Medicine, University of California Medical Center, San Francisco 94143, USA. egoetzl@itsa.ucsf.edu
Journal of immunology (UNITED STATES) Feb 15 1999, 162 (4) p2049-56, ISSN 0022-1767 Journal Code: IFB
Contract/Grant No.: HL31809, HL, NHLBI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Members of a subfamily of G protein-coupled receptors (GPCRs), encoded by five different endothelial differentiation genes (edgs), specifically mediate effects of lysophosphatidic acid (LPA) and sphingosine 1-phosphate (S1P) on cellular proliferation and differentiation. Mechanisms of suppression of apoptosis by LPA and S1P were studied in the Tsup-1 cultured line of human T lymphoblastoma cells, which express **Edg**-2 and **Edg**-4 GPCRs for LPA and **Edg**-3 and **Edg**-5 GPCRs for S1P. At 10-10 M to 10-7 M, both LPA and S1P protected Tsup-1 cells from apoptosis induced by Abs to Fas, CD2, and CD3 plus CD28 in combination. Apoptosis elicited by C6 ceramide was inhibited by S1P, but not by LPA, in part because ceramide suppressed expression of **Edg**-2 and **Edg**-4 surface receptors for LPA without affecting **Edg**-3 surface receptors for S1P. At 10-9 M to 10-7 M, LPA and S1P significantly suppressed cellular levels of the apoptosis-promoting protein Bax, without altering the levels of Bcl-xL or Bcl-2 assessed by Western blots and immunoassays. Transfections of pairs of **antisense** plasmids for **Edg**-2 plus **Edg**-4 and **Edg**-3 plus **Edg**-5, and hygromycin selection of transfectants with reduced expression of the respective **Edg** R proteins in Western blots, inhibited both protection from apoptosis and reduction in cellular levels of Bax by LPA and S1P. Thus, LPA and S1P protection from apoptosis is mediated by distinct **Edg** GPCRs and may involve novel effects on Bax regulatory protein.

2/3,AB/11 (Item 11 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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09589639 97459772 PMID: 9315732
The immediate-early gene product MAD-3/**EDG**-3/IkappaB alpha is an endogenous modulator of fibroblast growth factor-1 (FGF-1) dependent human endothelial cell growth.
Hla T; Zimrin AB; Evans M; Ballas K; Maciag T
Department of Physiology, University of Connecticut School of Medicine, Farmington 06030, USA. hla@sun.uchc.edu
FEBS letters (NETHERLANDS) Sep 8 1997, 414 (2) p419-24, ISSN 0014-5793 Journal Code: EUH
Contract/Grant No.: DK45659, DK, NIDDK; HL35627, HL, NHLBI; HL49094, HL, NHLBI
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The tumor promoter phorbol 12-myristic 13-acetate inhibits the growth of

inhibit its expression. The **antisense** IkappaB alpha PTO-treated cells exhibited an exaggera[redacted] growth response to fibrobl[redacted] growth factor-1 (FGF-1). In contrast, IL-1-induced growth arrest response was not modulated. These data suggest that the early response gene IkappaB alpha is an endogenous regulator of endothelial cell growth in vitro.

2/3,AB/12 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

1112e343 BIOSIS NO.: 199799747488
The immediate-early gene product MAD-3/**EDG**-3/I-kappa-B-alpha is an endogenous modulator of fibroblast growth factor-1 (FGF-1) dependent human endothelial cell growth.
AUTHOR: Hla Timothy(a); Zimrin Ann B; Evans Mark; Ballas Karin; Maciag Thomas
AUTHOR ADDRESS: (a)Dep. Physiol. MC3505, Univ. Conn. Sch. Med., 263 Farmington Ave., Farmington, CT 06030**USA
JOURNAL: FEBS Letters 414 (2):p419-424 1997
ISSN: 0014-5793
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The tumor promoter phorbol 12-myristic 13-acetate inhibits the growth of human endothelial cells and induces the formation of capillary-like, tubular structures. We report the novel growth regulatory function of the immediate-early gene, **edg**-3, which is identical to the I-kappa-B-alpha/MAD-3 gene. We employed phosphothioate oligonucleotides (PTO) directed against the translation initiation site of I-kappa-B-alpha to inhibit its expression. The **antisense** I-kappa-B-alpha PTO-treated cells exhibited an exaggerated growth response to fibroblast growth factor-1 (FGF-1). In contrast, IL-1-induced growth arrest response was not modulated. These data suggest that the early response gene I-kappa-B-alpha is an endogenous regulator of endothelial cell growth in vitro.

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? s edg and (antisens? or ribozym?)

            406  EDG
          29214  ANTISENS?
           5111  RIBOZYM?
     S1      19  EDG AND (ANTISENS? OR RIBOZYM?)
? rd

...completed examining records
     S2      12  RD (unique items)
? t s2/3,ab/all


 2/3,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11306555   21192228   PMID: 11150298
  Sphingosine 1-phosphate-induced endothelial cell migration requires the
expression of EDG-1 and EDG -3 receptors and Rho-dependent
activation of alpha vbeta3- and beta1-containing integrins.
  Paik JH; Chae Ss; Lee MJ; Thangada S; Hla T
  Center for Vascular Biology, Department of Physiology, University of
Connecticut Health Center, Farmington, Connecticut 06030-3501, USA.
  Journal of biological chemistry (United States)  Apr 13 2001,  276  (15)
  p11830-7,  ISSN 0021-9258  Journal Code: HIV
  Contract/Grant No.: DK-45659, DK, NIDDK
  Languages: ENGLISH
  Document type: Journal Article
  Record type: Completed
  Sphingosine 1-phosphate (SPP), a platelet-derived bioactive
lysophospholipid, is a regulator of angiogenesis. However, molecular
mechanisms involved in SPP-induced angiogenic responses are not fully
defined. Here we report the molecular mechanisms involved in SPP-induced
human umbilical vein endothelial cell (HUVEC) adhesion and migration.
SPP-induced HUVEC migration is potently inhibited by antisense
 phosphothioate oligonuclectides against EDG-1 as well as EDG-3
receptors. In addition, C3 exotoxin blocked SPP-induced cell attachment,
spreading and migration on fibronectin-, vitronectin- and Matrigel-coated
surfaces, suggesting that endothelial differentiation gene receptor
signaling via the Rho pathway is critical for SPP-induced cell migration.
Indeed, SPP induced Rho activation in an adherence-independent manner,
whereas Rac activation was dispensible for cell attachment and focal
contact formation. Interestingly, both EDG-1 and -3 receptors were
required for Rho activation. Since integrins are critical for cell
adhesion, migration, and angiogenesis, we examined the effects of blocking
antibodies against alpha(v)beta(3), beta(1), or beta(3) integrins. SPP
induced Rho-dependent integrin clustering into focal contact sites, which
was essential for cell adhesion, spreading and migration. Blockage of
alpha(v)beta(3) or beta(1)-containing integrins inhibited SPP-induced
HUVEC migration. Together our results suggest that endothelial
differentiation gene receptor-mediated Rho signaling is required for the
[illegible] alpha(v)beta(3) as well as beta(1)-containing
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DIALOG(R)File 155:MEDLINE(R)
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11306526 21192199 PMID: 11152468
Endothelial differentiation gene-2 receptor is involved in lysophosphatidic acid-dependent control of 3T3F442A preadipocyte proliferation and spreading.
Pages C; Daviaud D; An S; Krief S; Lafontan M; Valet P; Saulnier-Blache JS
INSERM U317, Institut Louis Bugnard, Universite Paul Sabatier, CHU Rangueil, Batiment L3, 31403, Toulouse cedex 04, France.
Journal of biological chemistry (United States) Apr 13 2001, 276 (15) p11599-605, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
EDG-2, **EDG**-4, **EDG** -7, and PSP24 genes encode distinct lysophosphatidic acid (LPA) receptors. The aim of the present study was to determine which receptor subtype is involved in the biological responses generated by LPA in preadipocytes. Growing 3T3F442A preadipocytes express **EDG**-2 and **EDG**-4 mRNAs, with no expression of **EDG**-7 or PSP24 mRNAs. Quantitative reverse transcriptase-polymerase chain reaction revealed that **EDG**-2 transcripts were 10-fold more abundant than that of **EDG**-4. To determine the involvement of the **EDG**-2 receptor in the responses of growing preadipocytes to LPA, stable transfection of **antisense EDG** -2 cDNA was performed in growing 3T3F442A preadipocytes. This procedure, led to a significant and specific reduction in **EDG** -2 mRNA and protein. This was associated with a significant alteration in the effect of LPA on both cell proliferation and cell spreading. Finally, the differentiation of growing preadipocytes into quiescent adipocytes led to a strong reduction in the level of **EDG**-2 transcripts. Results demonstrate the significant contribution of the **EDG** -2 receptor in the biological responses generated by LPA in 3T3F442A preadipocytes.

2/3,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11305893 21179099 PMID: 11134047
Sphingosine 1-phosphate protects human umbilical vein endothelial cells from serum-deprived apoptosis by nitric oxide production.
Kwon YG; Min JK; Kim KM; Lee DJ; Billiar TR; Kim YM
Department of Biochemistry, College of Natural Sciences, Department of Molecular and Cellular Biochemistry, School of Medicine, Kangwon National University, Chunchon, Kangwon-do 200-701, Korea. ygkwon@cc.kangwon.ac.kr
Journal of biological chemistry (United States) Apr 6 2001, 276 (14) p10627-33, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Sphingosine 1-phosphate (S1P) can prevent endothelial cell apoptosis. We investigated the molecular mechanisms and signaling pathways by which S1P protects endothelial cells from serum deprivation-induced apoptosis. We show here that human umbilical vein endothelial cells (HUVECs) undergo apoptosis associated with increased DEVDase activity, caspase-3 activation, cytochrome c release, and DNA fragmentation after 24 h of serum deprivation. These apoptotic markers were suppressed by the addition of S1P, the NO donor S-nitroso-N-acetylpenicillamine

enhancing Ca(2+)-sensiti[redacted] NOS activity without changes in the eNOS protein level. S1P-mediated ce[redacted] survival and NO productio[redacted]ere suppressed significantly by pretreatment with **antisense** oligonucleotide of **EDG**-1 and partially by **EDG**-3 **antisense**. S1P-mediated NO production was suppressed by the addition of pertussis toxin, an inhibitor of G(i) proteins, the specific inhibitor of phospholipase C (PLC), and the Ca(2+) chelator BAPTA-AM. These findings indicate that S1P protects HUVECs from apoptosis through the activation of eNOS activity mainly through an **EDG**-1 and -3/G(i)/PLC/Ca(2+) signaling pathway.

2/3,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11278181 21226767 PMID: 11278944
Two novel Xenopus homologs of mammalian LP(A1)/**EDG**-2 function as lysophosphatidic acid receptors in Xenopus oocytes and mammalian cells.
Kimura Y; Schmitt A; Fukushima N; Ishii I; Kimura H; Nebreda AR; Chun J
Department of Pharmacology, School of Medicine, University of California, San Diego, La Jolla, California 92093-0636, USA.
Journal of biological chemistry (United States) May 4 2001, 276 (18) p15208-15, ISSN 0021-9258 Journal Code: HIV
Languages: ENGLISH
Document type: Journal Article
Record type: In Process
Lysophosphatidic acid (LPA) induces diverse biological responses in many types of cells and tissues by activating its specific G protein-coupled receptors (GPCRs). Previously, three cognate LPA GPCRs (LP(A1)/VZG-1/**EDG**-2, LP(A2)/**EDG**-4, and LP(A3)/**EDG**-7) were identified in mammals. By contrast, an unrelated GPCR, PSP24, was reported to be a high affinity LPA receptor in Xenopus laevis oocytes, raising the possibility that Xenopus uses a very different form of LPA signaling. Toward addressing this issue, we report two novel Xenopus genes, xlp(A1)-1 and xlp(A1)-2, encoding LP(A1) homologs (approximately 90% amino acid sequence identity with mammalian LP(A1)). Both xlp(A1)-1 and xlp(A1)-2 are expressed in oocytes and the nervous system. Overexpression of either gene in oocytes potentiated LPA-induced oscillatory chloride ion currents through a pertussis toxin-insensitive pathway. Injection of **antisense** oligonucleotides designed to inhibit xlp(A1)-1 and xlp(A1)-2 expression in oocytes eliminated their endogenous response to LPA. Furthermore, retrovirus-mediated heterologous expression of xlp(A1)-1 or xlp(A1)-2 in B103 rat neuroblastoma cells that are unresponsive to LPA conferred LPA-induced cell rounding and adenylyl cyclase inhibition. These results indicate that XLP(A1)-1 and XLP(A1)-2 are functional Xenopus LPA receptors and demonstrate the evolutionary conservation of LPA signaling over a range of vertebrate phylogeny.

2/3,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11197951 21103260 PMID: 11160288
Lysophosphatidic acid receptor-selective effects on Jurkat T cell migration through a Matrigel model basement membrane.
Zheng Y; Kong Y; Goetzl EJ
Department of Medicine, University of California Medical Center, San Francisco, CA 94143, USA.
Journal of immunology (United States) Feb 15 2001, 166 (4) p2317-22,

Lysophosphatidic acid (LPA) and sphingosine 1-phosphate (S1P) from platelets and mononuclear phagocytes mediate T cell functions through endothelial differentiation gene-encoded G protein-coupled receptors (**Edg** Rs) specific for LPA (**Edg**-2, -4, and -7) or S1P (**Edg** -1, -3, -5, -6, and -8). Jurkat leukemic T cells with the SV40 virus large T Ag (Jurkat-T cells) express **Edg** -3>-2>-4 Rs, as assessed by RT-semiquantitative PCR and Western blots with anti-**Edg** R mAbs. Jurkat-T cells expressing predominantly **Edg**-2 R (Jurkat-T-2 cells) and **Edg**-4 R (Jurkat-T-4 cells) were developed by cotransfection with the respective sense plasmids and a mixture of **antisense** plasmids for the other **Edg** Rs, and hygromycin selection. Migration of Jurkat-T-4 cells, but not Jurkat-T-2 cells, through a layer of Matrigel on a 5-um pore polycarbonate filter was stimulated up to 5-fold by 10(-9) to 10(-6) M LPA and by 30-300 ng/ml of anti-**Edg**-4 R Ab, but not anti-**Edg**-2 R Ab. LPA and anti-**Edg** -4 R Ab also enhanced by up to 4-fold the expression of matrix metalloproteinase by Jurkat-T-4 cells, but not Jurkat-T-2 cells, as assessed by cleavage of [(3)H]-type IV human collagen in the Matrigel. Enhancement of matrix metalloproteinase-dependent trans-Matrigel migration of Jurkat-T cells by the chemokine RANTES was suppressed by anti-**Edg**-2 R Abs, but was stimulated by anti-**Edg** -4 R Abs. The opposite effects of **Edg**-2 and **Edg**-4 LPA receptors on trans-Matrigel migration and some other T cell functions provide receptor-selective mechanisms for regulation of T cell recruitment and immune contributions.

2/3,AB/6 (Item 6 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10825768 20428454 PMID: 10971577

Expression and characterization of **Edg** -1 receptors in rat cardiomyocytes: calcium deregulation in response to sphingosine 1-phosphate.

Nakajima N; Cavalli AL; Biral D; Glembotski CC; McDonough PM; Ho PD; Betto R; Sandona D; Palade PT; Dettbarn CA; Klepper RE; Sabbadini RA

Department of Biology and Heart Institute, San Diego State University, CA 92182-4614, USA.

European journal of biochemistry (GERMANY) Sep 2000, 267 (18) p5679-86, ISSN 0014-2956 Journal Code: EMZ

Contract/Grant No.: HL 63975, HL, NHLBI; NS/HL 25037, NS, NINDS

Languages: ENGLISH

Document type: Journal Article

Record type: Completed

Recent evidence indicates that sphingolipids are produced by the heart during hypoxic stress and by blood platelets during thrombus formation. It is therefore possible that sphingolipids may influence heart cell function by interacting with G-protein-coupled receptors of the **Edg** family. In the present study, it was found that sphingosine 1-phosphate (SphIP), the prototypical ligand for **Edg** receptors, produced calcium overload in rat cardiomyocytes. The cDNA for **Edg** -1 was cloned from rat cardiomyocytes and, when transfected in an **antisense** orientation, effectively blocked **Edg** -1 protein expression and reduced the SphIP-mediated calcium deregulation. Taken together, these results demonstrate that cardiomyocytes express an extracellular lipid-sensitive receptorsystem that can respond to sphingolipid mediators. Because the major source of SphIP is from blood platelets, we speculate that **Edg** -mediated SphIP negative inotropic and cardiotoxic effects may play important roles in acute myocardial ischemia where SphIP levels are probably elevated in response to thrombus.

10781532 20428654 PM[redacted] 10849424
Lipid phosphate phosphatase-1 and Ca2+ control lysophosphatidate signaling through **EDG**-2 receptors.
Xu J; Love LM; Singh I; Zhang QX; Dewald J; Wang DA; Fischer DJ; Tigyi G; Berthiaume LG; Waggoner DW; Brindley DN
Departments of Biochemistry (Signal Transduction Laboratories and Lipid Biology Research Group) and Cell Biology, University of Alberta, Edmonton, Alberta T6G 2S2, Canada.
Journal of biological chemistry (UNITED STATES) Sep 8 2000, 275 (36) p27520-30, ISSN 0021-9258 Journal Code: HIV
Contract/Grant No.: HL 61459, HL, NHLBI; RO1 61751, PHS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
The serum-derived phospholipid growth factor, lysophosphatidate (LPA), activates cells through the **EDG** family of G protein-coupled receptors. The present study investigated mechanisms by which dephosphorylation of exogenous LPA by lipid phosphate phosphatase-1 (LPP-1) controls cell signaling. Overexpressing LPP-1 decreased the net specific cell association of LPA with Rat2 fibroblasts by approximately 50% at 37 degrees C when less than 10% of LPA was dephosphorylated. This attenuated cell activation as indicated by diminished responses, including cAMP, Ca(2+), activation of phospholipase D and ERK, DNA synthesis, and cell division. Conversely, decreasing LPP-1 expression increased net LPA association, ERK stimulation, and DNA synthesis. Whereas changing LPP-1 expression did not alter the apparent K(d) and B(max) for LPA binding at 4 degrees C, increasing Ca(2+) from 0 to 50 micrometer increased the K(d) from 40 to 900 nm. Decreasing extracellular Ca(2+) from 1.8 mm to 10 micrometer increased LPA binding by 20-fold, shifting the threshold for ERK activation to the nanomolar range. Hence the Ca(2+) dependence of the apparent K(d) values explains the long-standing discrepancy of why micromolar LPA is often needed to activate cells at physiological Ca(2+) levels. In addition, the work demonstrates that LPP-1 can regulate specific LPA association with cells without significantly depleting bulk LPA concentrations in the extracellular medium. This identifies a novel mechanism for controlling **EDG**-2 receptor activation.

2/3,AB/8 (Item 8 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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10475226 20108368 PMID: 10645770
Induction of endothelial cell chemotaxis by sphingosine 1-phosphate and stabilization of endothelial monolayer barrier function by lysophosphatidic acid, potential mediators of hematopoietic angiogenesis.
English D; Kovala AT; Welch Z; Harvey KA; Siddiqui RA; Brindley DN; Garcia JG
Experimental Cell Research Program, The Methodist Research Institute, Clarian Health Partners, Inc., Indianapolis, IN 46202, USA.
Journal of hematotherapy & stem cell research (UNITED STATES) Dec 1999, 8 (6) p627-34, ISSN 1525-8165 Journal Code: DJX
Contract/Grant No.: PO1 HL 50864, HL, NHLBI; RO1 61751, PHS
Languages: ENGLISH
Document type: Journal Article
Record type: Completed
Angiogenesis, the formation of new blood vessels, is an important component of restoration of hematopoiesis after BMT, but the mediators involved in hematopoietic angiogenesis have not been identified. We